Vagal innervation modulates motor pattern but not initiation of canine gastric migrating motor complex

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Vagal innervation modulates motor pattern but not initiation of canine gastric migrating motor complex. Am J Physiol Gastrointest Liver Physiol 281: G283–G292, 2001.—To determine the role of vagal nerves in initiation and modulation of the gastric migrating motor complex (MMC), motor activity was recorded in four dogs before and after total abdominal vagotomy during fasting, after exogenous intravenous motilin and insulin, and after feeding. After vagotomy, a temporally coordinated cyclic gastric and small bowel MMC persisted with an unchanged period. During gastric phase III, vagotomy decreased number of contractions (42 ± 4 vs. 16 ± 2), number of groupings of contractions (14 ± 1 vs. 7 ± 1), and motility index (12 ± 1 vs. 10 ± 1) and increased the duration between groupings (1 ± 1 vs. 3 ± 1 min) (P < 0.05 in each). Before and after vagotomy, motilin and insulin induced a premature MMC with minor changes in contractile pattern. A 200-g liver meal but not a 50-g liver meal inhibited the gastric MMC after vagotomy. A cyclic MMC persisted after vagotomy, but the contractile pattern during gastric phase III was altered. After a short recovery period, vagal innervation to the stomach modulates the pattern but not the presence of gastric interdigestive motility during phase III.

truncal vagotomy; gastrointestinal motility; gastric phase III; motilin; Hollander test; pancreatic polypeptide

DURING FASTING, THE STOMACH and small intestine undergo a temporally coordinated cyclic motor pattern termed the migrating motor complex (MMC) (6, 26). Many studies have suggested important roles for hormones and other regulatory substances (2, 8, 17, 29), the enteric nervous system (19, 20), and extrinsic innervation (10, 22) in the control and modulation of this cyclic motility pattern.

The role of the vagus nerves in initiating and regulating this cyclic contractile activity, especially in the stomach, remains controversial. In 1975, Weisbrodt et al. (31) suggested, by studying dogs after total abdominal vagotomy, that vagal innervation plays little role in the organization of the interdigestive myoelectric complex of the small bowel. In 1977, Aeberhard et al. (1) studied the effects of proximal gastric vagotomy and total vagotomy in dogs and found that the vagal nerves were unimportant in the control of fasting activity. Our previous work in dogs showed the persistence of a cyclic motor pattern in the stomach after complete neural isolation of the stomach (30), staged neural isolation of the stomach (25), and upper gut neural isolation (23). In contrast, Diamant and colleagues (3, 9), in a series of studies utilizing acute, reversible cervical vagal blockade in dogs, have suggested that vagal innervation is necessary for initiation of the gastric MMC. The aim of this study was to attempt to more fully quantitate gastric interdigestive motor activity, especially the characteristics of phase III activity, before and after complete abdominal vagotomy. On the basis of our previous work (23, 25, 30), we hypothesized that, whereas vagal innervation is not necessary for initiation of the MMC or for the presence of phase III activity in the stomach, vagal innervation does modulate the contractile pattern and amount of contractile activity during phase III.

MATERIALS AND METHODS

Preparation of Animal Model

Surgical procedures and subsequent care and conduct of experiments were performed after approval from and according to criteria set forth by the Institutional Animal Care and Use Committee at Mayo Foundation in accordance with guidelines of the National Institutes of Health and Public Health Service Policy on the humane use and care of laboratory animals.

Four female mongrel dogs weighing 15–20 kg were anesthetized with intravenous methohexitol sodium (12.5 mg/kg; Brevital, Lilly, Indianapolis, IN); general anesthesia was maintained by inhalation of halothane. Through a midventral celiotomy, eight polyethylene manometry catheters (1.8 mm OD, 1.0 mm ID) were chronically inserted into the stomach, duodenum, and jejunum and fixed to the serosal surface. Four catheters were implanted into the gastric antrum 1, 2, 3, and 4 cm proximal to the pylorus, two into the duodenum, and two into the upper jejunum ~10 and 50 cm distal to the ligament of Treitz. Pyloromyotomy was performed to prevent the potential for gastric stasis after the total abdominal vagotomy to be performed at a second operation (see below). Manometry catheters were cemented within two stainless steel cannulas embedded in the abdominal wall. A modified Thomas gastric cannula was also fixed to the left abdominal wall to collect gastric juice for confir-
motilin (0.1 mg/kg) during fasting; the effects of injection of exogenous canine motilin at a dose of 0.1 µg/kg (Peninsula Laboratories, Belmont, CA), or after intravenous insulin at a dose of 0.7 U/kg, given 30 min after gastric phase III (>4 experiments per dog); and after the feeding of small (50 g) and large (200 g) pork liver meals (2 experiments each per dog) given 30 min after gastric phase III.

Plasma concentrations of motilin. Blood was collected on ice at the start of gastric phase III and at 30-min intervals thereafter. During each gastric phase III, a separate blood sample was drawn. In the digestive state, plasma concentrations of motilin were monitored at 30-min intervals beginning 30 min before feeding and until recurrence of gastric phase III after being given a 50-g pork liver meal. After the 200-g liver meal, blood samples were taken at 30-min intervals beginning 30 min before and until 240 min after feeding. Plasma was stored at −70°C for later batch assay by a well-established motilin immunoassay (23, 27).

Analysis of Data

Interdigestive experiments. All manometry recordings were analyzed by visual inspection for cyclic patterns of motor activity (i.e., the MMC). Criteria of the four phases of gastric and small bowel MMCs were used as in previous reports (6, 30). The mean duration of the MMC was assessed as the time between the start of successive phase III activity at one channel in each anatomic location (stomach, duodenum, jejunum). The mean durations of the individual phases of the MMC (I, II, III, and IV) at each of the three locations were also calculated. In an attempt to quantify gastric phase III activity, the following parameters were determined during each phase III activity by using two separate gastric manometry channels before and after vagotomy as shown in Fig. 1: 1) duration of gastric phase III; 2) maximum amplitude of contractions; 3) total number of contractions; 4) number of groupings of contractions; 5) mean number of contractions per grouping; 6) mean duration of individual groupings of contractions; 7) mean duration between groupings of contractions, and 8) motility index (MI) [calculated by computer analysis as MI = log (sum of amplitudes × frequency of pressure waves − 1), which is a function both of frequency and amplitude of individual contractions]. Also, the timing between the onset of the gastric and duodenal phase III was measured; positive values signify that the gastric phase III started before the duodenal phase III, whereas negative

![Fig. 1. Schema to explain quantification of gastric phase III.](image-url)
In contrast, after vagotomy, gastric pH remained. pH of gastric juice decreased to insulin injection. In conjunction with hypoglycemia, glycemia (40 mg/dl) was evident starting 15 min after exogenous insulin. Hypoglycemia induced by exogenous insulin led to the release of PP in the neuromuscular junction, and jejunal PP did not increase significantly. After vagotomy, plasma PP did not increase significantly.

Statistical Methods

Each experiment was performed at least four times per dog in the interdigestive state and two times per dog in the digestive state before and after vagotomy. Mean values for durations of the MMC, durations of the individual four phases of the MMC, and contractile parameters during gastric phase III were calculated on each experimental day, mean values across daily experiments were obtained in each dog, and finally grand means were calculated across all dogs. Differences before and after vagotomy were compared by using Student's t-test for paired data and repeated-measures ANOVA where appropriate, with P < 0.05 considered statistically significant. Values in the text are expressed as means ± SE.

RESULTS

Health of Dogs

All four dogs remained healthy before and after vagotomy. No dog lost >10% of body weight after either operation, and no dog vomited after vagotomy.

Vagal Integrity

There was a close relationship between blood glucose and pH of gastric juice after exogenous insulin. Hypoglycemia (40 mg/dl) was evident starting 15 min after insulin injection. In conjunction with hypoglycemia, pH of gastric juice decreased to <1.5 before vagotomy. In contrast, after vagotomy, gastric pH remained >2.0 despite the hypoglycemia. Hypoglycemia induced by exogenous insulin led to the release of PP in the neuromuscular junction.

Spontaneous Interdigestive Motor Patterns

A clearly visible, unmistakable cyclic motor pattern of contractile activity occurred in the stomach, duodenum, and jejunum in both neurally intact and vagotomized dogs. Motor activity showed an easily recognizable gastric MMC with four phases of activity both before and after vagotomy (Fig. 2, A and B). Table 1 quantitates interdigestive parameters of the cyclic activity. The period of the MMC and the duration of most phases of the MMC in the stomach, duodenum, and jejunum did not differ before and after vagotomy; however, phase I of the stomach in the vagotomized dogs was shorter than in neurally intact dogs, whereas phase II was longer (P < 0.05 each). Phase III activity in the stomach was temporally coordinated with phase III activity in the duodenum both before and after vagotomy. Every gastric phase III was closely associated temporally with a duodenal phase III that began simultaneously or just after the start of the gastric phase III. Timing between the onset of gastric and duodenal phase III was 6 ± 1 vs. 5 ± 2 min before and after vagotomy, respectively. In contrast, on occasion under our experimental conditions (prevagotomy 0 ± 0 and postvagotomy 8 ± 5%), a duodenal phase III was not preceded by a gastric phase III. After vagotomy, on occasion, a noncyclic pattern of continuous contractions occurred and persisted for 2–4 h in three of the four dogs despite persistent fasting and despite the presence of a typical MMC preceding this noncyclic pattern; this type of noncyclic “interdigestive” pattern was not seen in dogs before vagotomy. The incidence of this pattern was 22 ± 7% of the fasting recording studies (Fig. 3A).

Quantification of Gastric Phase III Motor Patterns

Table 2 compares the characteristics of gastric phase III activity before and after vagotomy in an attempt to quantitate the motor pattern as outlined in Fig. 1. Phase III activity occurred as a typical 10- to 30-min burst of high-amplitude contractions; the antral contractions during phase III occurred usually as multiple groups of two to four contractions separated by intervals of 30 s to 1 min. The overall duration and the maximum amplitude of contractions during phase III did not differ before and after vagotomy. In contrast, the total number of contractions, number of groupings of contractions, and MI during gastric phase III decreased after vagotomy, whereas the duration between groupings of contractions increased (each P < 0.05).

Plasma Concentrations of Motilin in the Interdigestive State

Motilin continued to cycle in close temporal coordination with the gastric MMC. Plasma motilin concentrations were greater during phase III than during phase I or II both before and after vagotomy (Table 3). Values before and after vagotomy did not differ. Plasma motilin concentrations cycled in temporal association with gastric phase III before the onset of the noncyclic motor pattern, but, during the period of this unique motor activity, motilin levels remained high and did not cycle (Fig. 3B).

Motor Patterns Induced by Exogenous Motilin or Insulin

Exogenous intravenous motilin (0.1 μg/kg) induced within 15 min a premature gastric phase III both before and after vagotomy in 81 ± 12% of the experi-
ments (Fig. 4, A and B). Phase III activity occurred in the stomach and then migrated aborad down the small bowel. The number of contractions, number of groupings of contractions, and MI during motilin-induced gastric phase III decreased, whereas the duration between groupings increased after vagotomy (Table 2). When compared within the same stage, the duration of motilin-induced gastric phase III was shorter than spontaneous phase III. When compared before vagotomy, the number of contractions, the number of group-

Table 1. Characteristics of interdigestive motility

<table>
<thead>
<tr>
<th></th>
<th>Before Vagotomy</th>
<th>After Vagotomy</th>
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<tbody>
<tr>
<td></td>
<td>Stomach</td>
<td>Duodenum</td>
</tr>
<tr>
<td>Duration of MMC</td>
<td>96 ± 4</td>
<td>97 ± 4</td>
</tr>
<tr>
<td>Duration of phase</td>
<td></td>
<td></td>
</tr>
<tr>
<td>I</td>
<td>58 ± 6</td>
<td>55 ± 8</td>
</tr>
<tr>
<td>II</td>
<td>17 ± 2</td>
<td>25 ± 4</td>
</tr>
<tr>
<td>III</td>
<td>19 ± 0</td>
<td>12 ± 1</td>
</tr>
<tr>
<td>IV</td>
<td>3 ± 2</td>
<td>5 ± 2</td>
</tr>
</tbody>
</table>

Values are means ± SE given in min for 4 dogs. MMC, migrating motor complex. *P < 0.05 compared with same parameter before vagotomy.
ings of contractions, and the MI during a premature gastric phase III were less than spontaneous phase III. However, these parameters were not different from spontaneous phase III after vagotomy except for the duration of gastric phase III. Timing between the onset of gastric and duodenal phase III did not differ before and after vagotomy (4 ± 1 vs. 3 ± 1 min).

After exogenous intravenous insulin (0.7 U/kg), a premature gastric MMC occurred in 63 ± 13% of the experiments before vagotomy, followed by continuous, noncyclic antral contractions for at least 60 min (Fig. 5A). After vagotomy, insulin was similarly successful (67 ± 13% of the experiments) in inducing a premature gastric and/or duodenal MMC, but the prolonged antral contractions afterward that were evident before vagotomy no longer occurred (Fig. 5B). The initiation of a premature phase III occurred 15–20 min after insulin and appeared to correlate with the development of hypoglycemia both before and after vagotomy. The timing between the onset of gastric and duodenal phase III before and after vagotomy differed somewhat (2 ± 2 vs. −4 ± 1 min; \(P < 0.05\)). Also, after vagotomy, the number of contractions, number of groupings of contractions, and the MI during the premature gastric phase III were decreased (\(P < 0.05\)). Before vagotomy, there were no differences between spontaneous and insulin-induced gastric phase III incidents. In contrast, after vagotomy, insulin-induced gastric phase III incidents were shorter in duration than spontaneous phase III incidents. In addition, there were many quantitative differences in the characteristics of

**Fig. 3.** A unique pattern of fasting motor activity in upper gastrointestinal tract after vagotomy. **A:** a constant, noncyclic contractile activity is evident after a typical gastroduodenal MMC in a representative dog. **B:** plasma motilin cycled with the gastric MMC before noncyclic activity but not during this noncyclic motor pattern in a representative dog on 1 day's experiment.
spontaneous and premature gastric phase III after vagotomy (Table 2).

**Postprandial Motor Patterns**

The duration of postprandial inhibition was similar before and after vagotomy with the 200-g meal (374 ± 34 vs. 298 ± 38 min; \( P > 0.05 \)). However, unlike before vagotomy, after vagotomy the 50-g meal did not inhibit the time to return of gastric MMC (218 ± 26 vs. 84 ± 19 min; \( P < 0.05 \)). The MI in the gastric antrum before vs. after vagotomy did not differ for the first hour after the feeding of the 200-g meal (9 ± 1 vs. 10 ± 1; \( P = 0.1 \)) or the 50-g meal (9 ± 1 vs. 10 ± 1; \( P = 0.5 \)).

**Plasma Concentrations of Motilin in the Digestive State**

After feeding the 200-g meal, plasma concentrations of motilin decreased and no longer cycled unless gastric phase III recurred before 240 min postprandially. Mean values during the postprandial state were 130 ± 15 and 135 ± 25 pg/ml before vagotomy and after vagotomy, respectively. In contrast, after the 50-g meal, plasma motilin began to cycle again when gastric phase III activity recurred. After the 50-g meal, mean values of plasma motilin concentrations during the postprandial period (before recurrence of phase III) were less than the plasma motilin concentrations at the time of return of the gastric phase III (before vagotomy: 155 ± 20 vs. 185 ± 15 pg/ml; after vagotomy: 145 ± 20 vs. 195 ± 15 pg/ml; \( P < 0.05 \) in each).

**DISCUSSION**

This study demonstrates that vagal innervation is not necessary for initiation of the gastric MMC nor for the temporal gastroduodenjejunal coordination in the interdigestive state. Vagal innervation does, however, modulate the contractile patterns during the gastric phase III. Not only exogenous motilin but also exogenous insulin induced a premature gastric MMC after spontaneous gastric MMC in the interdigestive state before and after vagotomy. Furthermore, plasma motilin concentrations cycled during the fasting state in accordance with gastric phase III after vagotomy. The 200-g liver meal, but not the 50-g meal, inhibited the onset of the gastric MMC after vagotomy.

The primary goal of this study was to show conclusively that vagal innervation to the canine stomach is not necessary for the initiation of spontaneous interdigestive activity. We quantitated gastric phase III activity not only by pattern analysis (visual inspection) but also by a novel method to quantitate multiple aspects of contractile activity during phase III (Fig. 1). We undertook this study primarily because previous work by Diamant and colleagues (3, 9) showed that the MMC in the canine stomach and duodenum was abolished during acute, reversible vagal blockade (cervical vagal cooling to 4°C); their work suggested that vagal innervation was necessary for initiation of the gastric component of phase III activity during fasting. In contrast, Gleystein et al. (7) showed that, when the vagal nerves were cooled intrathoracically just above the dia phragm, no effect on the presence of gastric phase III activity was evident, suggesting that vagal input to the stomach was not necessary for initiation of the gastric component of the MMC. This latter study by Gleystein et al. seems to have been largely overlooked.

Similarly, our previous work with models of staged nonvagal and subsequent vagal denervation of the stomach (25), complete, acute neural isolation of the stomach (30), complete neural isolation of the upper gut multivisceral complex (stomach, small bowel, pan-

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**Table 2. Quantification of gastric phase III**

<table>
<thead>
<tr>
<th></th>
<th>Before Vagotomy</th>
<th>After Vagotomy</th>
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<tbody>
<tr>
<td>Duration, min</td>
<td>19 ± 0</td>
<td>15 ± 2†</td>
</tr>
<tr>
<td>Maximum amplitude of contractions, mmHg</td>
<td>22 ± 3</td>
<td>13 ± 2†</td>
</tr>
<tr>
<td>No. of contractions</td>
<td>109 ± 18</td>
<td>86 ± 9</td>
</tr>
<tr>
<td>No. of groupings of contractions</td>
<td>14 ± 1</td>
<td>7 ± 1†</td>
</tr>
<tr>
<td>No. of contractions per grouping</td>
<td>3 ± 1</td>
<td>3 ± 1</td>
</tr>
<tr>
<td>Duration of groupings, min</td>
<td>1 ± 1</td>
<td>1 ± 1</td>
</tr>
<tr>
<td>Duration between groupings, min</td>
<td>1 ± 1</td>
<td>1 ± 1</td>
</tr>
<tr>
<td>Motility index</td>
<td>12 ± 1</td>
<td>11 ± 1†</td>
</tr>
<tr>
<td>Maximum amplitude of duodenal phase III, mmHg</td>
<td>72 ± 12</td>
<td>52 ± 8</td>
</tr>
<tr>
<td>Maximum amplitude of jejunal phase III, mmHg</td>
<td>70 ± 15</td>
<td>54 ± 10</td>
</tr>
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</table>

Values are means ± SE for 4 dogs. *P < 0.05 compared with same parameter before vagotomy. †P < 0.05 compared with spontaneous phase III in same stage.

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**Table 3. Plasma motilin concentrations during interdigestive state**

<table>
<thead>
<tr>
<th></th>
<th>Before Vagotomy</th>
<th>After Vagotomy</th>
</tr>
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<tbody>
<tr>
<td>Phase I</td>
<td>135 ± 15</td>
<td>150 ± 15</td>
</tr>
<tr>
<td>Phase II</td>
<td>120 ± 10</td>
<td>125 ± 15</td>
</tr>
<tr>
<td>Phase III</td>
<td>185 ± 15</td>
<td>195 ± 15</td>
</tr>
<tr>
<td>Phase IV</td>
<td>170 ± 15</td>
<td>185 ± 25</td>
</tr>
<tr>
<td>Noncyclic motor pattern?</td>
<td>215 ± 15</td>
<td></td>
</tr>
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</table>

Values are means ± SE given in pg/ml for (4 dogs). *Noncyclic activity did not occur before vagotomy but did after vagotomy in 3 of the 4 dogs; motilin concentrations did not cycle during this motor activity (see Fig. 5B). No differences noted between same phase before and after vagotomy. †P < 0.05 compared before and after vagotomy in same phase.
creas, liver, and proximal colon) (23), and most recently a staged nonvagal and subsequent vagal denervation of this multivisceral upper gut complex (28) all suggested that a cyclic gastric and small bowel interdigestive activity continued to occur after these models of selective and complete extrinsic denervation.

The present study was designed to reexamine this controversy with a straightforward canine model of a simple, supradiaphragmatic total abdominal vagotomy. By examining both subjective analysis of patterns (visual analysis Fig. 2, Table 1) and objective quantification of contractile patterns during phase III (Fig. 1, Table 2), we demonstrated quite clearly that a cyclic MMC in both stomach and upper small bowel continues after a total abdominal vagotomy (confirmed by a modified Hollander test) but that the pattern of contractions during the gastric phase III was altered; the number of contractions, number of groupings of antral contractions, and total motor activity (MI) were decreased, whereas the duration between groupings of contractions was increased. These objective findings strongly suggested that, whereas vagal innervation is neither necessary nor required for the presence or initiation of gastric interdigestive motility, vagal innervation does appear to modulate the pattern of contractions during fasting and after exogenous infusion of motilin.

How can we resolve the differences in the work by Diamant and colleagues (3, 9) and that of Gleysteen et al. (7) and ourselves (23, 25, 28, 30)? One potential explanation may be that the vagal nerves in the immediate supradiaphragmatic region contain some sympathetic fibers that may function as “inhibitory” nerves if unimpeded by vagal input (14, 16). Diamant’s group (5) extended their work by showing that the gastric MMC did not cycle when both the cervical vagal nerves were cooled and pharmacological adrenergic blockade was simultaneously established, again proposing the vagal nerves to be the most important factor for initiation of the gastric MMC. However, the concomitant “sympathetic” blockade was only a pharmacological adrenergic blockade, and it remains possible that nonadrenergic sympathetic neurotransmitters mediate an inhibitory response. Another explanation may be that acute reversible neural blockade (by cooling) has different effects than the chronic effects of complete surgical neural transection. The gut has a remarkable plasticity that allows an adaptation mediated in part through

![Fig. 4. Effect of exogenous IV motilin (0.1 μg/kg) in a representative neurally intact dog (A) and a representative vagotomized dog (B). In both stages, a premature gastric MMC occurred.](image-url)
the enteric nervous system. Indeed, Diamant and colleagues (4) have shown the ability of the stomach to regain a cyclic interdigestive activity many months after total duodenectomy, which we showed leads to the early loss of cyclic gastric interdigestive activity (27).

Our study also evaluated the effects of total abdominal vagotomy on interdigestive plasma concentrations of motilin and the motor response to exogenous motilin. We showed in the present study, as we have in other models of extrinsic vagal gastric denervation (24, 30), that plasma motilin concentrations cycle temporally with the gastric MMC, peaking during gastric phase III activity; also, when the interdigestive cyclic activity was disrupted, either spontaneously or after feeding, plasma motilin no longer cycled, peaks did not occur, and cyclic changes only recurred when the MMC recurred. Similarly, exogenous intravenous administration of motilin was able to induce a burst of gastric contractions in both the innervated and vagally denervated stomach followed by a characteristic intestinal MMC. Interestingly, the pattern of gastric contractions induced by motilin after vagotomy differed from that in the innervated stomach in ways similar to that of spontaneous gastric phase III. The number of contractions and the number of groupings of contractions in response to exogenous motilin were also fewer after vagotomy. However, these parameters were similar to the parameters of spontaneous gastric phase III in the vagally denervated stomach. Combined with the original work showing that plasma motilin cycles with the spontaneous MMC (12), exogenous intravenous motilin induces a “premature” MMC (11, 21), total duodenectomy inhibits the presence of a gastric MMC (27) at least for many months (4), and immunoneutralization of circulating plasma motilin disrupts the presence of the MMC in the gastroduodenal region (15), this present study supports the hypothesis that cyclic interdigestive activity in the stomach may be initiated and temporally coordinated with the small bowel MMC by cyclic changes in plasma concentrations of motilin.

We also investigated the role of the vagal nerves in mediating the motor effects of exogenous insulin at a dose great enough to induce hypoglycemia (0.7 U/kg). Before vagotomy, exogenous insulin induced a premature gastric and small bowel MMC, as shown by previous work (18), as well as a prolonged duration of antral contractile activity for at least 60 min. After
vagotomy, exogenous insulin continued to induce a premature gastrointestinal MMC, but the prolonged antral contractions no longer occurred. The gastric pattern of contractions, however, differed before and after vagotomy; insulin-induced gastric motor activity had fewer contractions and lesser amplitude as well as a fewer number of groupings of contractions and a lesser motility. Before vagotomy, insulin-induced gastric contractile patterns were similar to spontaneous contractions, but after vagotomy, the total gastric motility, as measured by MI, and the pattern of contractions (duration, maximal amplitude, number of contractions, number of groupings) decreased, suggesting a role of vagal innervation in mediating some of the effects of insulin on gastric contractile activity. However, vagal innervation to the stomach and the small bowel was not necessary for the ability of insulin to induce a premature MMC.

Finally, we showed that vagal innervation did play a role in postprandial inhibition of interdigestive gastrointestinal motility. After a small liver meal, vagotomy abolished the inhibition of the return of the MMC seen in the neurally intact dogs but did not alter the early motor effects, as measured by the MI for the first postprandial hour before the return of the MMC. In contrast, no differences in duration of inhibition of the MMC were noted after the larger 200-g meal. These observations suggest that the vagal nerves do mediate, in part, some of the postprandial motor events in the stomach and small intestine; however, other hormoneally mediated effects, as noted in the neurally isolated stomach (30) or after neural isolation of the entire upper gut complex (23) may play a dominant role with larger meals.

In conclusion, our study shows that vagal innervation to the stomach and small intestine is not necessary for the initiation or presence of the gastric or intestinal MMC, the cyclic changes in plasma motilin, or the motor response of a premature MMC after exogenous motilin or insulin. Vagal innervation does, however, modulate the pattern of gastric contractions during interdigestive activity and the postprandial duration of inhibition of the MMC after a small but not a larger oral meal.

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REFERENCES